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ORIGINAL ARTICLE

Non invasive adjustment of fluid status in critically ill patients on renal replacement therapy. Role of Electrical Cardiometry

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KEYWORDS

Electrical Cardiometry;
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Abstract *Background:* Electrical Cardiometry allows measurement of fluid status using thoracic fluid content (TFC), cardiac output, cardiac index, systemic vascular resistance index which could be ideal noninvasive hemodynamic monitoring for patients undergoing hemodialysis (HD). *Objectives:* Investigating the relation between changes in TFC and amount of fluid removal during HD session and to monitor hemodynamic parameters to avoid episodes of hemodynamic compromise during HD session. *Methods:* Thirty critically ill patients on HD were enrolled. Clinical assessment of volume overload and hemodynamics (BP, MAP, CVP), monitored by Electrical Cardiometry ICON® before HD and all through sessions. *Results:* Out of studied patients males represented 46.7% (n = 14) with mean age 48 ± 16 years. There was positive correlation between UF volume and TFC (r = 0.410, P = 0.025). Out of the 30 pts studied 18 pts (60%) were hemodynamically stable vs 12 pts (40%) that had hypotension represented by non responders group and had lower TFC compared to the hemodynamically stable group (26.45 kohm^{-1} vs 37.8 kohm^{-1}) with P value of 0.004 indicating that they were hypovolemic. Out of the 30 pts studied 18 pts (60%) weren't congested vs 12 pts (40%) remained persistently congested after accomplishing HD session with significantly higher TFC when compared to those who got rid of congestion (43.14 ± 9.9 kohm^{-1} vs 25.44 ± 5.5 kohm^{-1}) with P value of 0.0001 indicating that they were still hypervolemic. Using analysis of ROC curve TFC at 25.34 kohm^{-1} was a significant predictor of hypotension with P value of 0.002, AUC 83.4%, sensitivity 67% and specificity 100%. Also TFC cutoff value predicting persistent congestion was 37.02 kohm^{-1} with P value of 0.0001, AUC 95.8%, sensitivity 83% and

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specificity 100%. *Conclusion:* Electrical Cardiometry is an evolving noninvasive tool for adjusting fluid status of critically ill patient on RRT using thoracic fluid content as an indicator of fluid status that could be used to avoid hemodynamic instability and persistent volume overload and congestion during and after HD session.

1. Introduction

Intradialytic hypotension and orthostatic hypotension after the procedure are significant and independent risk factors affecting mortality in dialysis patients [1].

Several noninvasive methods for hemodynamic monitoring and determination of fluid status of critically ill patients have been developed. That can aid the intradialytic assessment of fluid removal objectively and avoid hemodynamic instability [2].

Impedance cardiography (ICG) and Electrical Cardiometry (EC) are recently developed technologies to measure thoracic fluid content (TFC), cardiac output (CO) and other hemodynamic parameters. Both ICG and EC derive CO from measurements of Thoracic Electrical Bioimpedance (TEB) [3].

TEB is the electrical resistance to high frequency low amplitude current that is transmitted from electrodes placed on the upper and lower thorax. The resultant value is indirectly proportional to the volume of thoracic fluids such that increasing fluid in the thorax results in less TEB. Therefore, the inverse of TEB, and thus changes in CO, are reflected as a change in total bioimpedance or fluid conductivity [3].

One of the parameters examined is thoracic fluid content (TFC), which is inversely associated with the patient's transthoracic electrical bioimpedance, and reflects the total (intravascular and extravascular) fluid volume contained in the chest cavity [4].

The fluid content is a pronouncedly variable parameter of a human's chest and this is why the dynamic measurements of chest impedance by means of ICG can reliably and accurately reflect its alterations. Potential changes in thoracic fluid content are directly proportional to total fluid changes; thus, ICG and EC parameters can prove to be extremely significant for the monitoring of thoracic blood volume changes during hemodialysis (HD) session [5].

The aim of our study was to investigate the relation between changes in the TFC and the amount of ultra filtration fluid volume and to provide a means of easily tracking fluid status during hemodialysis, to help in adjusting fluid removal (rate and amount). Secondly to continuously monitor hemodynamic parameters with the ultimate goal of understanding how monitoring patients with this system can help to avoid episodes of clinically significant hemodynamic compromise.

2. Patients and methods

Our study was designed as a cross-sectional study that was conducted between June 2014 and March 2015 in the critical care medicine department of Cairo university hospitals.

The study enrolled thirty critically ill patients on renal replacement therapy admitted because of renal failure (acute or chronic), AND/OR fluid overload due to cardiac or hepatic causes. Patients less than 18 years old, with implantable cardiac pace maker or defibrillator, significant valvular lesions and pleural effusion, pregnant females, end stage hepatic, cardiac or pulmonary diseases, terminal malignancy and patients refused to participate in the study were excluded.

The study was approved by our local scientific and ethics committee. All enrolled patients had signed an informed consent for participation in the study and subjected to detailed history taking and thorough clinical examination for clinical signs of volume overload including congested neck veins, orthopnea, rales on chest auscultation, and lower limb edema. Weighing the patient before and after HD session, CVP measurement before and after HD session and SBP, DBP and MAP measurement every 30 min, full laboratory investigations including blood gases, electrolytes and renal function, 12 lead ECG recordings and routine echocardiography examination including assessment of systolic function by 2D imaging, Doppler and M mode function, measurement using ATL machine 33 with 3.5 mHz probe.

Chest X-ray examination for detection of pulmonary congestion, abnormal chest X-ray was defined as having abnormal fluid (pulmonary congestion) if they were graded 1, 2 or 3 Table 1 [6,7].

All patients were monitored during HD session using new model "EC" based device (ICON®) starting 15–30 min prior to HD and every 30 min thereafter, with a 15 to 30 min stabilization period after the termination of HD session.

The Electrical Cardiometry monitor (Electrical Cardiometry monitor, ICON® Cardiotronics, Inc.) was connected to the sensor cable and the patient data were fed. The ICON® monitor incorporates an algorithm which transforms the ohmic equivalent of mean aortic blood flow acceleration into an equivalent of mean aortic blood flow velocity.

The ICON® device emits a high frequency (50 kHz) and low-amperage (2 mA) alternating electrical current of constant amplitude via a pair of surface electrodes across the left side of the thorax. The voltage drop due to the current application is

Table 1 Chest radiography grading definitions [7].

Garde	Chest X-ray grading
Grade 0	Normal pulmonary vascular distribution
Grade 1	Stage 1 pulmonary venous hypertension: vascular redistribution due to hypoxia induced basilar vasoconstriction from non visualized early edema
Grade 2	Stage 2 pulmonary venous hypertension: vascular redistribution due to early "peribronchial cuffing" or late "Kerly's B line" interstitial edema
Grade 3	Stage 3 pulmonary venous hypertension: vascular redistribution and perihilar pulmonary edema

registered together via a second pair of sensing electrodes, which were located at the left side of the neck and the left side of the thorax at the level of the xiphoid process, inside the current electrodes. Prior to opening of the aortic valve, the red blood cells assume a random orientation (there is no blood flow in the aorta). After aortic valve opening, the pulsatile blood flow forces the red blood cells to align in parallel with the blood flow, the change from random orientation to alignment of red blood cells upon opening of aortic valve generates a characteristic steep increase of conductivity or dZ (t) (corresponding to a steep decrease of impedance) beat to beat [8].

The following Hemodynamic Parameters measured by ICON® thoracic fluid content (TFC), cardiac index (CI) by body surface area CI (BSA) and by weight CI (WT), mean arterial pressure (MAP), systemic vascular resistance (SVR) and systemic vascular resistance index (SVRI) by body surface area SVRI (BSA) and weight SVRI (WT), these parameters will be measured before starting HD session by 15 min (zero reading 0) after 30 min (1st reading), after 1 h (2nd reading), after 2 h (3rd reading), after 3 h (4th reading), after the end of the session of HD by 15–30 min (5th reading).

The patients were subjected to intermittent HD sessions using Hemodialysis Machine, FRESENIUS 4008S. Nephrology staff according their policy tailored the protocol of HD session.

Data of HD session were recorded including blood flow (BF) pump, blood flow (BF) pump change, UF rate, UF volume, and complications during HD.

- The study population were further subdivided into two groups, responders and non-responders as guided by the clinical assessment of volume overload or Hemodynamic stability:
  - o *Responders* were defined as those who were not congested at the end of HD session as guided by clinical assessment of signs of congestion mentioned previously or remained hemodynamically stable.
  - o *Non-responders* those who were still congested at the end of HD session or developed hypotension (a decrease in SBP $\geqslant$ 20 mmHg or MAP $>$ 10 mmHg) and/or associated with symptoms that were relieved by cessation of ultra filtration or HD session, decrease blood pump flow rate and/or improved with administration of bolus saline.

Statistical analysis was done using Statistical Package for Social Sciences (SPSS) software, 22 license for WindowsTM (SPSS Inc., Chicago, Illinois). Quantitative variables were described using mean ± standard deviation (SD) if they were normally distributed. Categorical variables were described using frequencies and percentages. Bivariate analysis of categorical variables was done using Chi Square test with Yates Continuity correction for 2×2 tables. Whenever cell frequency was less than five, Fisher's Exact test was used. Comparing two groups of quantitative variable was done using Independent-Samples Student t test for parametric data, and Mann–Whitney test for non-parametric one. The correlation between two quantitative variables was explored using Pearson test for parametric data and Spearman's test for non-parametric one. In all cases, the 2-sided significance was always taken as p value, and a p value less than 0.05 was considered statistically significant.

3. Results

Our study included thirty critically ill pts, who had IHD session in the critical care medicine department. The age distribution of the study population ranged between 21 and 74 years (mean 48.5 ± 16.5). 53.3% of the study groups were females while males represented 46.7%. Out of 30 patients, 12 (40%) pts were still congested at the end of the HD session and 12 (40%) pts experienced hypotension and were defined as non responders, there was no statistically significant difference regarding demographic and co-morbid conditions in the study groups with $P\ value > 0.05$ Table 2.

In our study, we found that, TFC before HD sessions was statistically correlated with CI (BSA) 0 before HD session with $r = 0.410$ and $P = 0.025$, as well CVP before HD session with $r = 0.546$ and $P = 0.003$ Fig. 1.

There was a significant positive correlation between TFC5 and CI (BSA) 5 after HD session with $r = 0.557$ and $P = 0.001$, CVP after HD session with $r = 0.389$, $P = 0.041$ Fig. 2.

There was a significant positive correlation between TFC change (ΔTFC) with CI (BSA) 5 after HD session with $r = 0.484$, $P = 0.007$, while it was negative correlation with MAP0 before HD session with $r = -0.394$, $P = 0.031$, SVR 5 after HD session with $r = -0.434$, $P = 0.016$ and SVRI (BSA) 5 with $r = -0.389$, $P = 0.05$ Fig. 3.

Similarly, % ΔTFC had nearly same correlation as ΔTFC. We found a statistically significant positive correlation between UF volume and TFC ($r = 0.40$, $P = 0.029$), ΔTFC ($r = 0.429$, $P = 0.018$), % ΔTFC ($r = 0.387$, $P = 0.035$), while there was no statistically significant correlation with CI, SVR, SVRI $P > 0.05$.

Table 2 Study population characteristics.

	All ($n = 30$)	Responders ($n = 18$)	Non responders ($n = 12$)	P value
Age (years)	48.5 ± 16.5	47.94 ± 16.53	49.25 ± 17.3	0.837
Gender (M)	14 (46.6%)	8 (26.6%)	6 (20%)	1.0
Smoking	9 (30)%	6 (20%)	3 (10%)	.704
DM	14 (46.7%)	8 (26.7%)	6 (20%)	1.000
HTN	19 (63.3%)	13 (43.3%)	6 (20%)	.395
ARF	25 (83.3%)	13 (43.3%)	12 (40%)	.066
CRF	5 (16.7%)	6 (20%)	0 (0%)	.057
CHD	4 (13.3%)	1 (3.3%)	3 (10%)	.274
Com CLD	6 (20%)	4 (13.3%)	2 (6.7%)	1.000
Chest inf.	11 (36.7%)	4 (13.3%)	7 (23.3%)	.063
DCL	4 (13.3%)	1 (3.3%)	3 (10%)	.274
SLE	4 (13.3%)	3 (10%)	1 (3.3)	.255
Sepsis	6 (20%)	5 (16.7%)	1 (3.3%)	0.358

M: male, DM: diabetes mellitus, HTN: hypertension, ARF: acute renal failure, CRF: chronic renal failure, CHD: coronary heart disease, Com CLD: compensated chronic liver disease, inf.: infection, DCL: disturbed conscious level, SLE: systemic lupus.

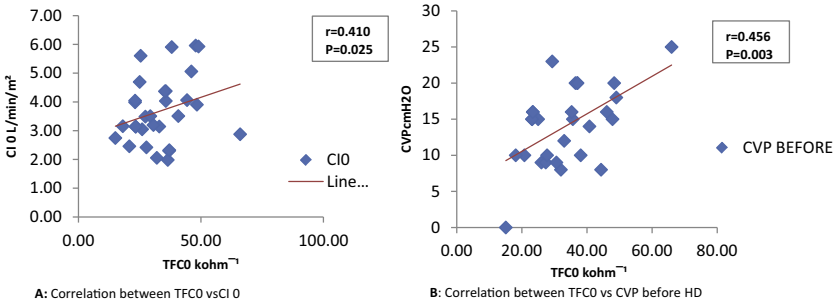

Figure 1 Correlation between TFC before HD and (A) CI0, (B) CVP before HD.

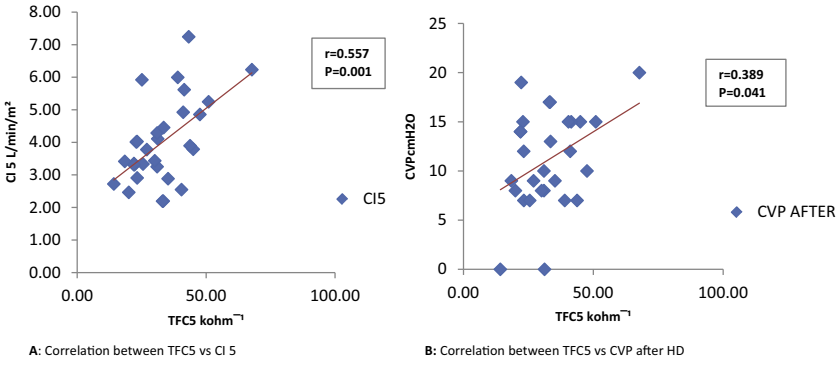

Figure 2 Correlation between TFC after HD and (A) CI5, (B) CVP after HD session.

We thoroughly analyzed pts who had persistent congestion, we found there was a statistically significant difference between responder and nor responders regarding congested NVs and pulmonary congestion, congestion in CXR and normal CXR $P < 0.05$. Mean CVP dropped from mean of 14 ± 5.4 cm H_2O before HD session to mean of 11.14 ± 5 cm H_2O after HD session. Similarly weight dropped from mean of 71.9 ± 16.7 kg before HD session to mean of 71.03 ± 16.44 kg after HD session, with no statistically significant difference in study groups $p > 0.05$.

There was no statistically significant difference between responders and non responders regarding UF volume and rate, blood flow (BF) pump rate and change ($p \geqslant 0.05$) as shown in Table 3.

There was a statistically significant difference regarding TFC 0, 5, average TFC and % ΔTFC ($p < 0.05$), while there was no statistically significant difference regarding ΔTFC ($p > 0.05$), as shown in Table 4.

Also, there was a statistically significant difference between responders and non responders regarding CI 0, 5 (BSA) and average CI (BSA) with $p \leqslant 0.05$. There was no statistically significant difference between responders and non responders regarding MAP, SVR and SVRI (BSA) with $p > 0.05$.

ROC curves were plotted to determine the cutoff value of TFC to predict persistent congestion Fig. 4, which was 35.66 kohm^{-1} at the beginning of HD session with P value of 0.0001, AUC 91.2%, sensitivity 88.9% and specificity 83.3%, Also TFC at the end of HD session was 34.38 kohm^{-1} with P value of 0.0001, AUC 98.6%, sensitivity 91% and specificity 100%, while average TFC cutoff value was 37.02 kohm^{-1} with P value of 0.0001, AUC 95.8%, sensitivity 83% and specificity 100%.

We thoroughly analyzed pts who had hypotension, we found that, there was no statistically significant difference between responders and nor responders regarding congested NVs and pulmonary congestion, LL edema $P > 0.05$. Mean CVP dropped from mean of 14 ± 5.4 cm H_2O before HD session to mean of 11.14 ± 5 cm H_2O after HD session. Similarly weight dropped from mean of 71.9 ± 16.7 kg before HD session to mean of 71.03 ± 16.44 kg after HD session, with no statistically significant difference in study groups with $p > 0.05$. There was a statistically significant difference between responders and non responders regarding blood flow (BF) pump rate and BF pump change ($p = 0.002$ and 0.0001) respectively while there was no significant difference between responder and non responders regarding UF volume and rate ($p > 0.05$) Table 5.

There was a statistically significant difference between responders and non responders regarding TFC 0, 5 and average TFC ($p < 0.05$), while there was no statistically significant difference regarding ΔTFC and % ΔTFC ($p > 0.05$), as shown in Table 6.

Also there was a statistically significant difference between responders and non responders regarding MAP 5 and average MAP with $p < 0.05$, whereby there was a drop of MAP values in non responders group from 92 ± 16.7 mmHg to 83 ± 17.2 mmHg. In contrast MAP in responders group was changed during HD session then returned to the same value at beginning of the HD session (97.3 ± 1.31 mmHg, 94.5 ± 10.7 then 97.3 ± 11.4 mmHg at the end). There was no statistically significant difference between responders and

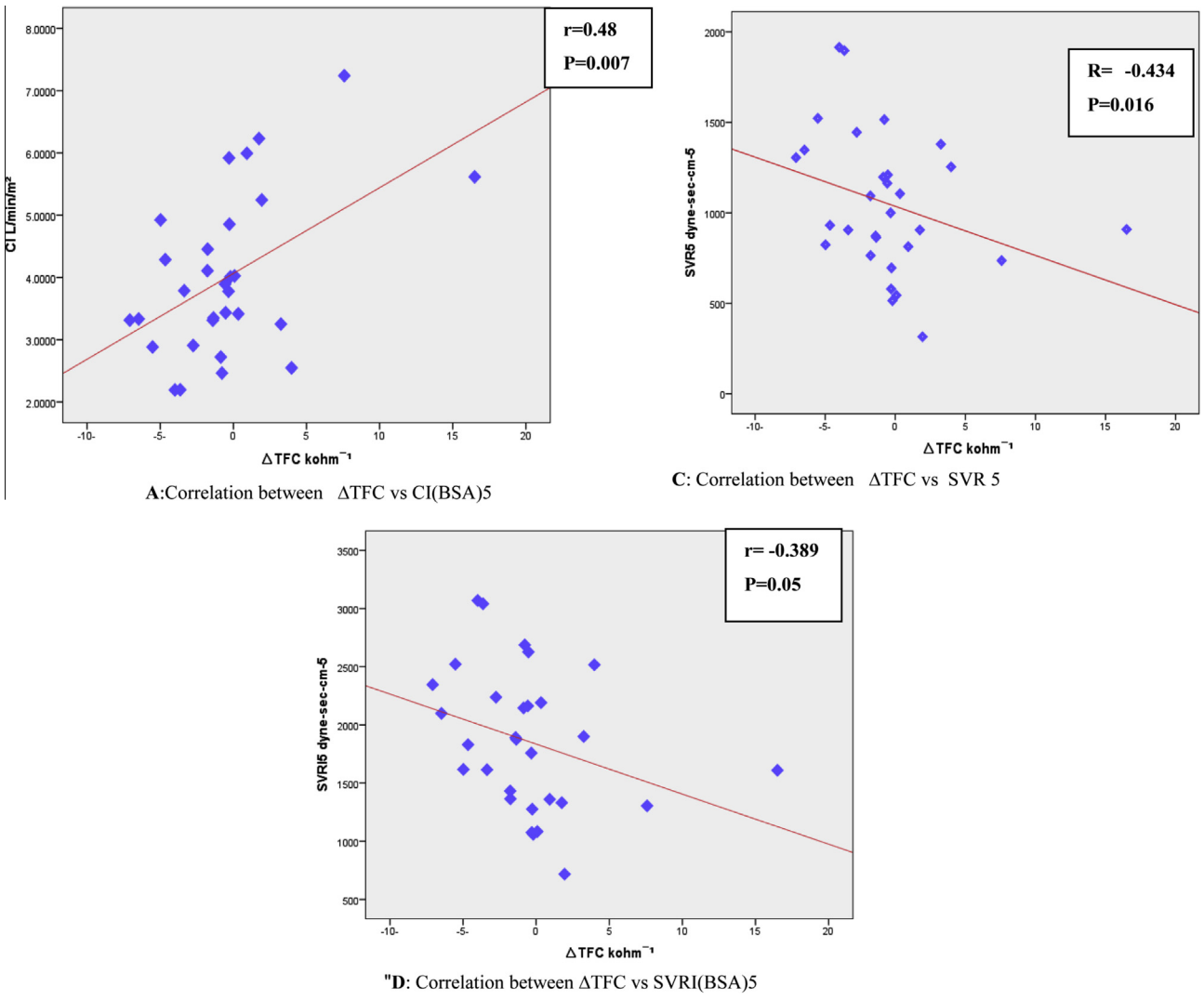

Figure 3 + ve correlation between ΔTFC and CI after HD (A), −ve correlation between ΔTFC before HD, SVR and SVRI after HD (B and C).

Table 3 Comparison between responders and non responders according to HD parameters.

HD parameters	Responders	Non responders	*P* value
	Mean ± SD	Mean ± SD	
UF volume ml/session	1500 ± (1237)	1917 ± (1311)	0.329
UF rate ml/h	479 (415)	575 ± (340)	.513
BF Pump rate ml/h	248 ± (36)	229 ± (33)	.169
	No. & %	No. & %	
BF pump change	5 (17.7%)	2 (6.7%)	0.66

non responders regarding CI (BSA), SVR and SVRI (BSA) with $p > 0.05$.

ROC curves were plotted to determine the cutoff value of TFC to predict hypotension Fig. 5, which was 26.6 $kohm^{-1}$ at the beginning of HD session with *P* value of 0.001, AUC 85%, sensitivity 94.4% and specificity 75%. Also TFC at the end of HD session was 26.2 $kohm^{-1}$ with $P = 0.01$, AUC 78.2%, sensitivity 88.9% and specificity 75%, while average TFC cutoff value was 25.34 $kohm^{-1}$ with *P* value of 0.002, AUC 83.4%, sensitivity 67% and specificity 100%.

Table 4 Comparison between responders and non responders in relation to TFC by ICON®.

TFC by ICON	Responders		Non responders		*P* value
	Mean	±SD	Mean	±SD	
TFC0	27.24	6.46	42.55	10.34	0.0001
TFC5	25.44	5.57	43.90	9.14	0.0001
Average TFC	26.67	6.32	43.14	9.49	0.0001
ΔTFC	−1.805	2.561	1.349	6.043	0.058
% ΔTFC	−5.90	8.23	5.72	21.20	0.044

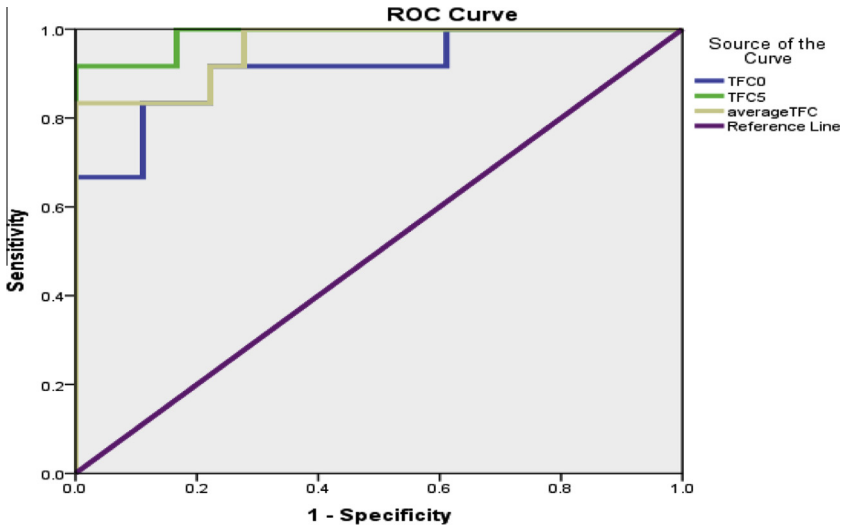

Figure 4 ROC curve for prediction of persistent congestion.

Table 5 Comparison between responders and non responders according to HD parameters.

HD parameters	Responders		Non responders		*P* value
	Mean	±SD	Mean	±SD	
UF rate ml/h	605.83	431.43	384.17	260.99	0.12
UF volume ml/session	1972	1334	1208	1033	0.11
BF Pump rate ml/hr	225	25.72	263.25	37.51	0.002
	No.	%	No.	%	
BF Pump change	0	0	7	23.3	0.0001

Table 6 Comparison between responders and non responders in relation to TFC by ICON®.

TFC by ICON	Responders		Non responders		*P* value
	Mean	±SD	Mean	±SD	
TFC0	38.09	9.86	26.29	9.12	.003
TFC5	36.69	10.52	27.03	11.07	.023
Average TFC	37.80	9.90	26.45	9.67	.004
ΔTFC	−1.41	3.53	0.75	5.56	.203
% ΔTFC	−3.75	10.08	2.50	21.45	.291

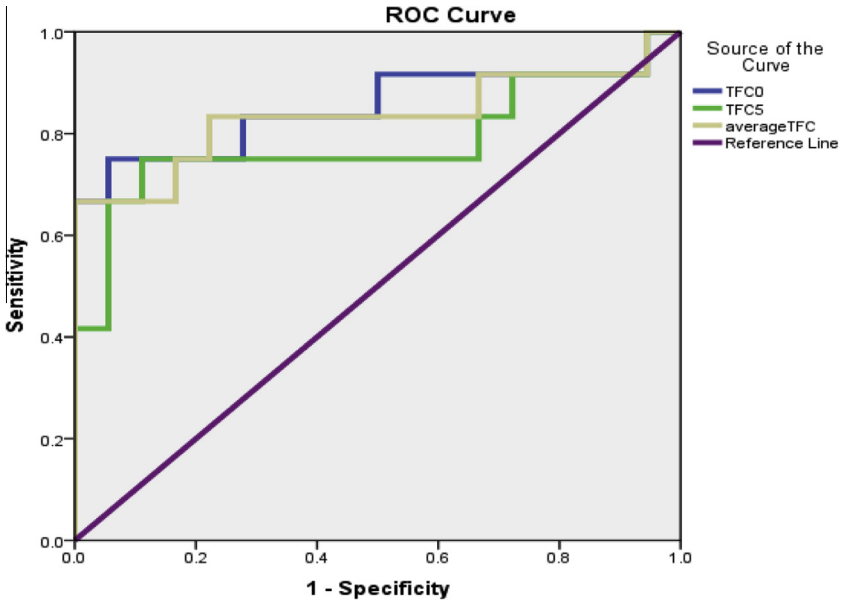

Figure 5 ROC curve for prediction of hypotension.

4. Discussion

Decisions during hemodialysis (HD) in critically ill patients are usually challenging to determine the proper amount of fluid should be filtered to unload while avoiding intradialytic hypotension (IH). The evaluation of fluid status is generally approached from clinical observation of body weight change, congestion, edema, blood pressure and chest X-ray. However, evaluation on clinical grounds alone is not accurate enough in HD patients. For this reason, more objective methods, such as biochemical markers, bioimpedance analysis (BIA) and inferior vena cava (IVC) diameters have been developed for the assessment of fluid status. However, no single method has emerged as a gold standard, and the combination of these methods is generally needed to complement their respective limitations [9].

Electrical Cardiometry (EC) is a recently developed technology to measure the cardiac output. Both ICG and EC derive CO from measurements of Thoracic Electrical Bioimpedance (TEB) [3].

There is a real need to refine FR based on a real-time indication of how much of the patient's water is being removed [10].

Our study showed a positive correlation between UF volume and TFC, these findings were in agreement with Wynne et al. [5], who studied TFC using non invasive ICG in CRF patients undergoing hemodialysis and Kossari et al. [11], De Nicola and Sucre [12], who studied TFC using non invasive ICG (NICCOMO®, Medis, Germany) in critically ill patients during CVVHDF proved the correlation between TFC and FR., reported similar results using a new tool bioreactance.

An animal study by van de Water et al. [13], showed that, in 10 similarly anesthetized dogs overhydrated with intravenous saline TFC closely correlated with the UF ($r = 0.93$). We could not expect such a good value, considering that TFC has a different range of normal values for both men and women.

Positive statistical agreement between TFC with CVP before and after HD session was spotted in Piccoli et al. [14], who found a positive correlation between CVP and bioimpedance measurement in critically ill patients more than total body water measured by bioimpedance vector analysis as well as highlighted by our data.

Moreover, Ebert et al. [15] found a significant linear correlation between the alterations of central venous pressure and the alterations of the thoracic baseline bioimpedance. Hence, TFC should be a good indicator of fluid status over time and might help guide the need for and extent of FR during HD, at least in each individual patient.

In our study 40% of the study population developed hypotension as defined previously (a decrease in SBP $\geqslant$ 20 mmHg or MAP $>$ 10 mmHg).

This percentage agreed with previous studies that showed an incidence of intradialytic hypotension ranging between 15% and 60% of various studies done by Wynne et al. [5], Magdy et al. (40–60%) [16], John et al. (45–60%) [17], Pavan et al. (18%) [18], Straver et al. (37.5%) [19]. The different populations studied in prior studies that enrolled only CRF patients could explain the discrepancy in the results.

Our results regarding (ICON®) parameters showed that there was a statistically significant difference regarding TFC in the subgroups studied, with lower TFC values in non responders group compared to responders (26.45 kohm^{-1} vs 37.8 kohm^{-1}) indicating that they were hypovolemic. Also there was no statistically significant difference regarding TFC change (ΔTFC) between the 2 groups. The drop of BP was transient in the responder group and persistent in non responders at the end of the dialysis session with no effect on SVR.

In our study we found that there was a statistically significant difference regarding MAP between responders and non responders, whereby there was a drop of MAP values in the non responders group from 92 ± 16.7 mmHg to 83 ± 17.2 mmHg. In contrast MAP in the responders group was changed during HD session then returned to the same

value at the beginning of HD session (97.3 ± 1.31 mmHg–94.5 ± 10.7 then 97.3 ± 11.4 mmHg at the end).

In our study there was no statistically significant difference regarding SVR, SVRI (BSA, WT), which were however higher and dropped more in the non responders group.

This was in agreement with Straver et al. in 1998 [19], who reported intradialytic hypotension during a deep fall in SVR with a concomitant increase in heart rate. Although less pronounced, CO and BV in the hypotensive group also decreased more than in the stable group. Coritsidis et al. in 2003 [20], found that changes in SVRI and HR were associated with hypotension in diabetics undergoing HD [5]. Ahmed et al. in 2003 [21], found that 28 of 37 patients undergoing HD experienced a BP drop and that, in 20 of these 28, a drop in SVR occurred, resulting in clinical instability [5].

In our study 40% of the study population had persistent congestion at the end of HD session. Also, Our study results showed that there was no statistically significant difference between responders and non responders regarding lower limb edema with $P > 0.05$. This was in agreement with Agarwal et al. [22], who found that edema is of limited value in diagnosing excess intravascular volume.

Our study results showed that there was a statistically significant difference between responders and non responders regarding pulmonary congestion and congested neck veins ($P < 0.05$). This was supported by Wang et al. in 2005 [23], who performed a meta-analysis of 18 studies that evaluated the utility of the history, physical examination, and diagnostic tests in diagnosing HF and volume overload in patients presenting to the emergency department with dyspnea. Among all presenting symptoms, paroxysmal nocturnal dyspnea was most helpful, followed closely by orthopnea and peripheral edema.

They concluded that clinical examination is a poor predictor of the patient's volume status and fluid resuscitation should proceed cautiously, even in patients who are clinically hypovolaemic.

According to Eisenberg et al. [24], when clinicians were asked to predict hemodynamic parameters based only on history and physical examination, their performance was poor, In this study, pulmonary artery occlusive (wedge) pressure was correctly predicted only 30% of the time. CO, SVR, and RAP were correctly predicted approximately 50% of the time.

In our study there was a statistically significant difference between responders and non responders regarding pulmonary congestion in CXR with $P < 0.05$ with more congestion in non responders.

In agreement with this finding, Milzman et al. [6], who conducted a study in the heart failure population to determine if there was a relationship between thoracic bioimpedance and chest radiograph changes. Their data suggested a linear relationship between the grading of the severity of the CXR in relation tointrathoracic fluid volume, and the changes of mean thoracic thoracic bioimpedance.

According to Peacock et al. in 2000 [7], there was no linear relationship between a worsening radiographic appearance and thoracic bioimpedance. They suspected that this was due to the poor sensitivity of CXR for detecting pulmonary fluid in heart failure patients. The radiographs of chronic heart failure patients may not show "congestive" signs and may actually have relatively normal CXR results despite excess thoracic volume. In chronic heart failure patients admitted to an ED with shortness of breath, thoracic bioimpedance determination may be superior to CXR interpretation in determining fluid state and planning an appropriate treatment plan.

Our results regarding (ICON®) parameters showed that there was a statistically significant difference regarding TFC between subgroups with higher TFC values in non responders compared to responders ($43.14 \pm 9.9\ \text{kohm}^{-1}$ vs $25.44 \pm 5.5\ \text{kohm}^{-1}$) indicating that they were still hypervolemic.

We assumed that the high TFC values at baseline and at end of HD session would be anticipated in volume-overloaded patients in need of more UF volume to improve the clinical condition of the pts.

This finding was supported with the PREDICT study in 2006 [25], in which the correlation between stroke volume and the values of TFC appeared to bear substantial prognostic significance for patients with congestive heart failure.

Our study found that there was a statistically significant difference regarding CI (BSA, WT), where there were higher CI (BSA) values in the non responders group than responders, also CI (WT) values were higher in non responders.

In our study we investigated the predictive value of TFC in relation to persistent congestion at end of HD session in HD pts as an indicative of the fluid status of HD pts to guide the need for ultrafiltration and to tailor UF goal accordingly. ROC curves were plotted to determine the cutoff value of TFC to predict persistent congestion.

To conclude our discussion, there was a correlation between TFC measured by ICON®, UF volume and volume overload after HD sessions, hence it could be an objective monitoring parameters to guide fluid management and ultra filtration in critically ill patients. Also suggested cutoff values of TFC could be usefully used as an objective tool to avoid hemodynamic instability in association with other hemodynamic parameters measured by ICON and to tailor individual fluid management of HD patients.

In conclusion hemodynamic instability is still a common challenging complication during HD patients that contribute to CVD risk as well as volume overload after HD session as evident by electrical bioimpedance and clinical assessment.

Electrical bioimpedance using a new modified module ICON® is an evolving non-invasive tool for adjusting fluid status of critically ill patients on RRT using thoracic fluid content (TFC) as an indicator of fluid status along with other hemodynamic parameters that could be used to avoid hemodynamic instability and persistent volume overload and congestion during and after HD session. We recommend doing larger randomized interventional studies to set clear protocols for UF volume based on an objective tool to improve patients' outcome.

4.1. Study limitations

The small sample size is one of the limitations of our study and lacking an objective gold standard for determining dry weight which mainly depends on clinical assessment for that we aim to present an objective mean to guide fluid management with future larger studies with a strict protocol for HD patients to improve outcome and decreasing CVD morbidities.

Our study is a cross sectional observation study that lacks intervention to adjust the UF volume based on our bioimpedance measurements which were taken during one session of

HD, and decisions were left for nephrologists to changes HD parameters.

We didn't use an invasive hemodynamic technique to compare with our results, we could answer this question as our study wasn't planned to validate the technique, rather aimed to demonstrate the applicability of electrical bioimpedance derived parameters to guide fluid and hemodynamic management in HD critically ill patients. Also there were many validation studies of the used technique that were compared to the invasive TD technique.

Conflict of interest

Authors declare that there is no conflict of interest.

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